HEMODYNAMICS OF LEFT VENTRICULAR APEX-AORTIC VALVED CONDUITS

J. RICHARD SHANEBROOK, PH.D., AND MATHEW L. LEVINE, B.S.

A flow model for analyzing the fluid mechanics of left ventricular-aortic valved conduits has been established. The model is based on a parallel flow circuit analogy of Ohm's law, the classic analysis of Gorlin and Gorlin[1] for the determination of valvular areas, and an empirical constant, introduced by Gentle,[2] that is descriptive of prosthetic heart valve performance. Favorable comparisons with clinical data indicate that the flow model is capable of predicting volumetric rates of flow through the valved conduit and through the aorta. Applications of the model are discussed in terms of altering the design of the valved conduit to improve its performance. The effect of valvular efficiency on conduit performance is investigated, and it is concluded that the Starr-Edwards ball valve and the Hancock 250 valve offer attractive alternatives if the objective is to increase the volumetric rate of flow through the valved conduit, or to decrease the volumetric rate of flow through the stenotic aortic valve, or both.

In certain cases of left ventricular outflow tract obstruction, surgeons have provided relief to patients by implanting a prosthetic valved conduit extending from the apex of the left ventricle to the aorta as shown schematically in Figure 1. This provides a second outflow tract for the flow exiting from the left ventricle and leaves the natural outflow tract intact. As described by Cooley and Norman,[3] and Bernhard, Poirier, and LaFarge,[4] this surgical technique has proven useful in cases of congenital or acquired supravalvular, valvular, and subvalvular stenoses where more conventional approaches such as aortic valvotomy or commissurotomy are expected to produce less than optimal results, due to the severity of the obstruction or because of its reoccurrence. This concept of a double outlet ventricle appears to have originated in 1910 with Alexis Carrel who constructed left ventricular-aortic shunts with vein grafts. In more recent years, prosthetic conduits with valves have been employed clinically, and the interested reader is referred to a recent review of this subject by Norman, Nihill and Cooley.[5]

From the Department of Mechanical Engineering, Union College, Schenectady, New York 12308.

Address for reprints: J. Richard Shanebrook, Ph.D., Union College, Schenectady, New York 12308.

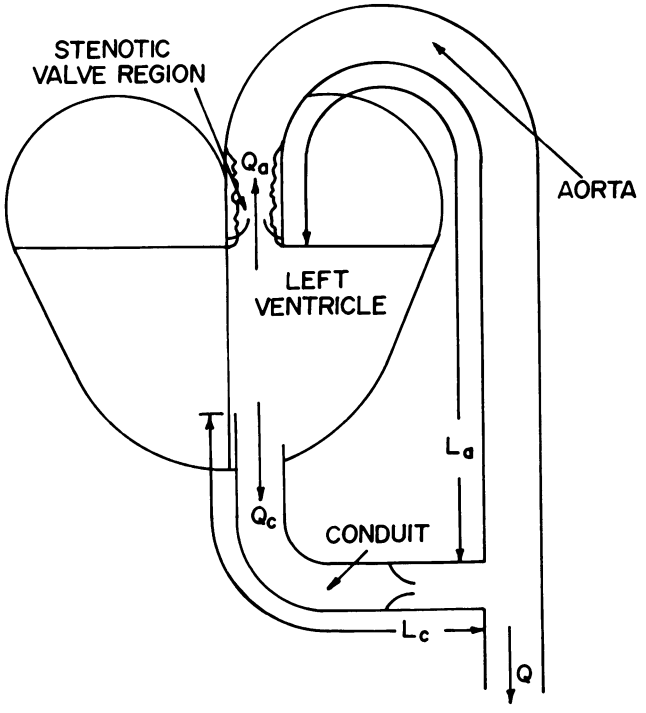

Fig. 1 Schematic diagram of prosthetic valved conduit extending from the left ventricle to the aorta.

The purpose of this report is to analyze the fluid mechanics of valved conduits that provide a parallel flow network for the movement of blood from the left ventricle to the aorta. This analysis will result in a flow model that will be compared with clinical data and then used to investigate how various design parameters may be changed to alter the performance of the valved conduit.

THE FLOW MODEL

The theoretical approach developed here was motivated by the work of Furuse and associates[6] who analyzed the hemodynamics of vein grafts when used as conduits to bypass obstructions in coronary arteries. Figure 2 is a schematic diagram of this situation, which shows a vein graft bypassing an obstruction in a coronary artery. Furuse et al[6] showed that Poiseuille's law for steady, laminar flow of a Newtonian fluid through a straight, cylindrical tube can be used to determine the volumetric rate of flow, Q_g, through the vein graft. Writing this law in the form,

$$\frac{\Delta P}{Q} = \left(\frac{128\mu}{\pi}\right)\frac{L}{D^4}, \qquad (1)$$

the right side of this equation may be interpreted as the resistance to flow, R, as discussed by Burton.[7] Thus,

$$Q = \frac{\Delta P}{R}, \tag{2}$$

which is analogous to Ohm's law. This analogy permits flow circuits to be analyzed in a manner similar to that for direct current (DC) electrical circuits. For example, Figure 3 is the flow circuit corresponding to Figure 2, and from the theory for parallel circuits, it follows that the total volumetric rate of flow through the circuit is given by

$$Q = Q_g + Q_o = \frac{\Delta P}{R_g} + \frac{\Delta P}{R_o + R_{ca}} = \frac{\Delta P}{\frac{1}{R_g} + \frac{1}{(R_o + R_{ca})}},$$

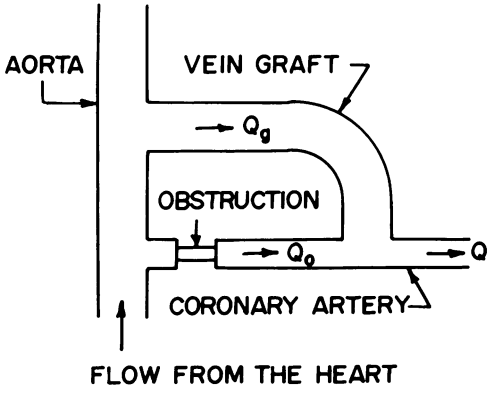

Fig. 2 Schematic diagram of vein graft bypassing an obstruction in a coronary artery.

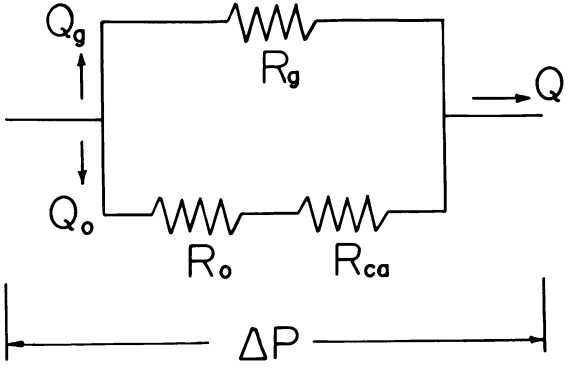

Fig. 3 Flow circuit corresponding to the schematic diagram of Figure 2.

where subscripts g, o, and ca refer to the graft, obstruction, and coronary artery, respectively.

For the case considered here (shown schematically in Figure 1), the corresponding flow circuit is shown in Figure 4 where Q_a and Q_c represent the volumetric rates of flow through the aorta and conduit, respectively. From circuit theory it then follows that

$$Q_a = \frac{\Delta P}{R_a + R_{av}},$$

$$Q_c = \frac{\Delta P}{R_c + R_{cv}},$$

and

$$\frac{Q_a}{Q_c} = \frac{R_c + R_{cv}}{R_a + R_{av}}, \tag{3}$$

where the last equation is the ratio of antegrade (aortic flow) to retrograde (conduit flow).

The resistances R_c and R_a, in equation (3), may be determined from the right side of equation (1). Thus,

$$R_c = \left(\frac{128\mu}{\pi}\right)\frac{L_c}{D_c^4} \tag{4}$$

and

$$R_a = \left(\frac{128\mu}{\pi}\right)\frac{L_a}{D_a^4} \tag{5}$$

The valvular resistances, R_{av} and R_{cv}, in equation (3) may be determined from the classic analysis of Gorlin and Gorlin[1] who found for the valve cross-sectional area,

$$A = C_1 \frac{Q}{\sqrt{\Delta P}}, \tag{6}$$

where Q is volumetric flow rate and ΔP is the loss in pressure across the valve. This result was established by a steady, inviscid flow analysis for flow through an ideal orifice. Introducing valve diameter into the left side of equation (6) and squaring each side gives,

$$\Delta P = C \frac{Q^2}{D^4}, \tag{7}$$

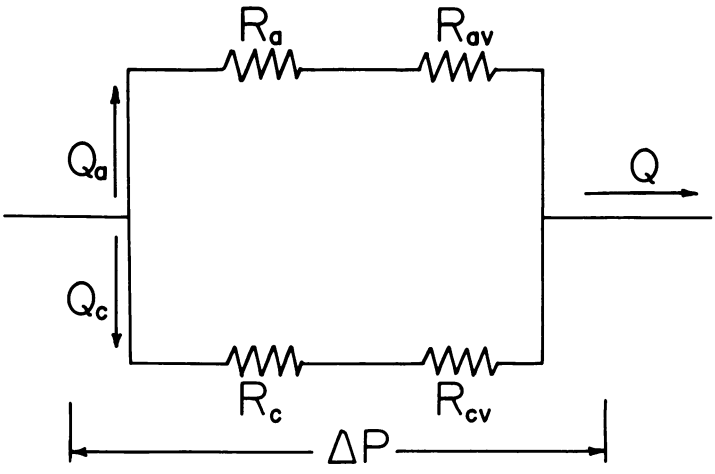

Fig. 4 Flow circuit corresponding to the schematic diagram of Figure 1.

which is a form of the Gorlin and Gorlin formula found useful by Gentle[2] in evaluating the efficiency of prosthetic heart valves. Gentle[2] also established that the constant, C, in equation (7) has the value 1,775 (mm Hg) (mm^4) (min^2) per $liter^2$ for flow through an ideal orifice in which there is no friction or jet contraction and where ΔP is expressed in millimeters of mercury, Q in liters per minute and D in millimeters. Further, Gentle[2] was able to show that experimental data for a given prosthetic heart valve when plotted as ΔP vs Q^2/D^4 forms a nearly linear relationship. Thus, for a given valve design, the constant C in equation (7) represents the slope of this line, and when compared to the value for an ideal orifice, can be used to establish an efficiency for the valve. This also provides a convenient measure of the relative performance of different prosthetic heart valve designs since slopes and efficiencies can be easily compared.

For the present analysis, equation (7) can be used to determine prosthetic valvular resistance, since

$$R = \frac{\Delta P}{Q} = C \frac{Q}{D^4}, \tag{8}$$

where C is the slope of the line corresponding to the particular prosthetic heart valve under consideration.

Equations (3), (4), (5), and (8) provide the basis for determining the relative volumetric flow rates through the aorta and valved conduit. Comparison with clinical data provides a means for evaluating the accuracy of this flow model.

COMPARISON WITH CLINICAL DATA

Norman, Nihill and Cooley[5] have presented clinical results for five patients in sufficient detail to enable comparison with the flow model of the previous section. The data are summarized in Table I. In addition to these data, it was necessary to secure information on the conduit diameter used in each patient, an estimate for the tissue annulus diameter of the aortic valve, and the valve characteristics for the conduit and aortic valves.

Conduit diameters for the patients listed in Table I were specified by Johnson[8] and are listed in Table II.

An estimate for the tissue annulus diameter of the aortic valve was obtained by assuming that this dimension is approximately equal to the diameter of the ascending aorta just distal to the sinuses of Valsalva. This assumption is based on information given by Swanson and Clark.[9] Data for the size of the ascending aorta as a function of age has been reported by Dittmer and Grebe[10] from which values for the aortic diameter can be calculated. These values are listed in Table II. It is noted that the value for patient WB was obtained by interpolation, and it was assumed that after age 40 the aorta does not increase in size. Unfortunately, it appears that the data reported by Dittmer and Grebe[10] was obtained from cadavers and therefore the aorta was not subjected to the usual intraaortic pressure. To account for the distensibility of the aorta, a correction factor was established by considering the results reported by Swanson and Clark[9] for aortic diameter as a function of pressure.

Series 7 and 8 of the data reported by Swanson and Clark[9] give aortic diameters for average pressures from zero mm Hg to 100 mm Hg. The latter value was assumed to be representative of average aortic pressures under resting conditions. For these two series, the increases in aortic diameter from zero to 100 mm Hg were 37% and 33%, respectively, or an average increase of 35%. With this average increase, the tissue annulus diameters in Table II were adjusted upward to the values shown in the column, "Adjusted Tissue Annulus Diameter."

TABLE I. Clinical Results Reported by Norman, Nihill and Cooley[5] for Five Patients

Patient Initials	Age (Years)	Aortic Flow (% Cardiac Output)	Conduit Flow (% Cardiac Output)
WB	10	71	29
JH	7	59	41
VM	15	57	43
GM	36	68	32
EH	72	60	40
Mean		63	37
Standard Deviation		±6	±6

TABLE II. Conduit and Aortic Dimensions for the Patients Listed in Table I.

Patient Initials	Conduit Diameter (mm)	Aortic Diameter (mm)	Adjusted Tissue Annulus Diameter (mm)
WB	14.0	15.0	20.2
JH	14.0	13.0	17.6
VM	16.0	16.8	22.7
GM	18.0	20.0	27.0
EH	20.0	20.0	27.0

The prosthetic valve employed by Norman, Nihill and Cooley[5] in each of the conduits was a Hancock prothesis of appropriate size. It was further learned by contacting Hancock Laboratories[11] that these valves were Hancock Model 100 prostheses consisting of a model 242 valve fastened inside a synthetic graft. From the in vitro data reported by Wright[12] for this valve, it was possible to construct the graph shown in Figure 5, where D is the sewing ring or tissue annulus diameter. It is noted that a prosthetic valve presents two possible diameters for use in equation (8). These are the inner (orifice) diameter and the sewing ring (tissue annulus) diameter. It was found to be more convenient to employ the tissue annulus diameter in

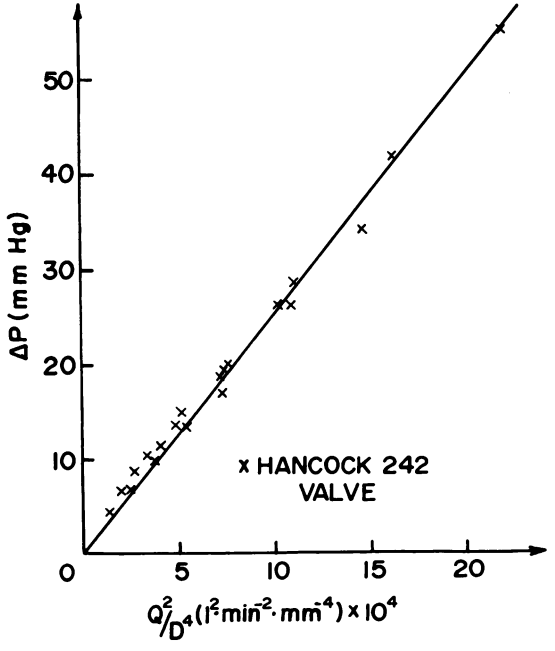

Fig. 5 Plot of experimental data reported by Wright[12] for the Hancock 242 prosthetic heart valve.

equation (8) for analyzing the flow through conduit valves (for example, the tissue annulus diameter is the same as the conduit diameter). For the results shown in Figure 5, the constant C was determined to be 25,300 (mm Hg) (mm^4) (min^2) per $liter^2$ with a standard least squares fit analysis (y – intercept forced through zero).

Since data such as those shown in Figure 5 for prosthetic valves are not available for stenotic human aortic valves, it is necessary to make a number of assumptions concerning the flow characteristics of stenotic aortic valves. Following the reasoning of Gentle,[2] it is assumed that the efficiency of a healthy, natural aortic valve is 100%. That is, it is assumed that equation (8) describes the flow through the normal aortic valve where C has the value 1,775 (mm Hg) (mm^4) (min^2) per $liter^2$ and D is the diameter of the orifice area formed by the three aortic leaflets in the open position during forward flow. It thus becomes convenient to adopt the inner (orifice) diameter when analyzing the flow through a natural aortic valve with equation (8) instead of the annulus diameter which was adopted for the prosthetic valve. It is further assumed that these same conditions prevail when the valve becomes stenotic. That is, it is assumed that the valve still has the same constant of 1,775 (mm Hg) (mm^4) (min^2). The only difference is that the orifice diameter, D, has been reduced due to the stiffer valve leaflets. This assumption can be reasoned on the grounds that the flow through the valve senses only the area of the orifice opening and not the structural changes within the tissues of the valve leaflets. It remains, then, to determine the orifice diameter of the stenotic valves. For this, the concept of critical stenosis as discussed by Strandness and Sumner[13] is useful. Critical stenosis is defined here as the percentage by which the cross-sectional area of a vessel must be reduced in order to produce a measureable drop in blood flow. Strandness and Sumner[13] indicate that critical stenosis is dependent on flow rate. For example, at high flow rates a stenosis of 60% produces an appreciable pressure drop, whereas at low flow rates the critical stenosis may be 90% to 95%. Data obtained from the iliac arteries of anesthetized dogs indicate that there is no reduction of flow until the stenosis reaches 80%. Since the flow conditions considered here involve patients at rest, this latter value for moderate flow rates will be adopted. Further support for this assumption is provided by the results of Bellhouse and Bellhouse who developed a mathematical theory for the flow through a stenotic aortic valve. They compared their theory with in vitro flow data obtained with a model stenotic aortic valve and reported that the best agreement was found for a stenosis of 78.8%.

The above-stated conditions and assumptions provide sufficient information to determine the resistance to flow due to the stenotic aortic valve and the conduit valve. The resistances given by equations (4) and (5) for the conduit and aorta, respectively, are determined in the Appendix where it is demonstrated that these resistances are relatively small when compared to the valvular resistances. Thus, equation (3) reduces to

$$\frac{Q_a}{Q_c} = \frac{R_{cv}}{R_{av}} \tag{9}$$

Where R_{cv} and R_{av} are given by equation (8). That is,

$$R_{cv} = C_{cv} \frac{Q_c}{D_c^4} \tag{10}$$

and

$$R_{av} = C_{av} \frac{Q_a}{D_{av}^4} \tag{11}$$

Combining with equation (9) gives,

$$\frac{Q_a}{Q_c} = \frac{C_{cv}}{C_{av}} \left(\frac{Q_c}{Q_a}\right) \frac{D_{av}^4}{D_c^4},$$

which can be simplified by rearranging and taking the square root of each side. Thus,

$$\frac{Q_a}{Q_c} = \left(\frac{C_{cv}}{C_{av}}\right)^{1/2} \left(\frac{D_{av}}{D_c}\right)^2 \tag{12}$$

The diameter of the aortic valve, D_{av}, can be related to the tissue annulus diameter by recalling that a stenosis of 80% leaves 20% of the cross-sectional area of the aorta available for flow through the open aortic valve. Thus,

$$\frac{\pi D_{av}^2}{4} = 0.20 \left(\frac{\pi D_{ta}^2}{4}\right)$$

which gives,

$$D_{av}^2 = 0.20\, D_{ta}^2$$

Combining with equation (12) and introducing $C_{av} = 1{,}775$ gives,

$$\frac{Q_a}{Q_c} = 0.20 \left(\frac{C_{cv}}{1{,}775}\right)^{1/2} \left(\frac{D_{ta}}{D_c}\right)^2, \tag{13}$$

which is the final result needed to compare calculated flow rates with those reported by Norman, Nihill and Cooley.[5]

Table III presents a comparison between the clinically measured aortic and conduit flow rates, as a percent of cardiac output, with corresponding values determined from equation (13) where values for D_{ta} and D_c were obtained from Table II and $C_{cv} = 25{,}300$ from Figure 5. Overall, the agreement between theory and the clinical results for these five patients is good. However, it is noted that the theory overpredicts conduit flow rates in four of the five cases analyzed. This could be due to the natural flow patterns created in the left ventricle during ventricular filling. That is, the mitral valve generates a vortex motion within the ventricle which acts to orient the flow towards the natural outflow tract. The present model does not account for this effect. Another factor is that during late systole, Cooley et al[15] observed in one patient that the left ventricular apex essentially occluded the entrance to the conduit. This effect is not accounted for by the present model and, if present in the cases treated here, it would cause the percentage of aortic flow to be greater than that predicted.

APPLICATION OF THE FLOW MODEL

Having established the validity of the flow model, given by equation (13), for the five clinical cases presented by Norman, Nihill and Cooley,[5] it is of interest to investigate how certain physical characteristics of the valved conduit could be altered in order to improve performance. For example, the results in Table III indicate that the valved conduit carries less than half of the total flow rate in all five patients, with the mean value being only 37% of the measured cardiac output. It is evident from equation (13) that the amount of conduit flow could be increased by employing a conduit valve with a higher efficiency (lower value for C_{cv}) or by increasing the diameter of the conduit, or both. Due to anatomical restrictions, it is probably more practical to consider replacing the Hancock 242 prosthesis with a more efficient valve. For this reason four other valves were evalu-

TABLE III. Comparison Between Clinically Measured Aortic and Conduit Flow Rates and Theoretical Values from Equation (13)

	Clinical Results		Theoretical Results	
Patient Initials	Aortic Flow (% Cardiac Output)	Conduit Flow (% Cardiac Output)	Aortic Flow (% Cardiac Output)	Conduit Flow (% Cardiac Output)
WB	71	29	61	39
JH	59	41	54	46
VM	57	43	60	40
GM	68	32	63	37
EH	60	40	58	42
Mean	63	37	59	41
Standard Deviation	±6	±6	±4	±4

TABLE IV. Valve Constant, C_{cv}, for Homograft, Björk-Shiley Tilting Disc, Hancock 250, and Starr-Edwards Caged Ball Valves

Valve	C_{cv} (mm Hg • mm^4 • lit^{-2} • min^2)
Homograft	
Forrester et al[16]	4,900
Björk-Shiley tilting disc	
Björk[17]	11,100
Hancock 250	
Wright[12]	13,300
Starr-Edwards caged ball	
Forrester et al[16]	17,000

ated regarding their possible performance in a valved conduit for left ventricular-aortic shunts.

Table IV presents the results found for the valve constant, C_{cv}, for four other valves that are possible candidates for a valved conduit. These results were obtained by plotting experimental data, available in the references listed in Table IV, in the form of Figure 5 and then by linear regression determining the valve constant. By way of example, patient EH was selected to illustrate the effect that each of these valves would have on the amount of aortic and conduit flows if they were employed in the valved conduit. These results are summarized in Table V, where it is seen that a homograft valve would have the greatest effect on conduit flow by increasing it from 43% to 63% of cardiac output. The Hancock 250 valve predicts an even distribution between conduit and aortic flow rates. All four of the valves listed in Table IV predict higher conduit flow rates than the Hancock 242 valve.

Although these results indicate that a homograft valve is best in terms of increasing conduit flow, such valves are not readily available. Also, the

TABLE V. Effect of Valve Selection on Aortic and Conduit Flow Rates for Patient EH as Determined from Equation (13)

Valve	Aortic Flow (% Cardiac Output)	Conduit Flow (% Cardiac Output)
Homograft	38	62
Björk-Shiley tilting disc	48	52
Hancock 250	50	50
Starr-Edwards caged ball	53	47
Hancock 242	58	42

Björk-Shiley tilting disc valve was reported by Cooley et al[15] to produce unsatisfactory results when used in this manner because of late thrombosis of the valve. Thus, the better choices for a future conduit valve may be the Starr-Edwards ball valve or the Hancock 250 valve if increased conduit flow rates are desired. This, of course, raises an important question as to whether or not elevated conduit flow rates would be detrimental to the systemic circulation. Cooley and Norman[3] temporarily occluded the ascending aorta in most of their patients to determine the effect upon the systemic circulation. They reported that after a brief fall in blood pressure of approximately 20%, the systemic pressure rose to a normal level, indicating that the conduit could, if necessary, carry the entire cardiac output. Moreover, Cooley and Norman[3] mention a case of aortic insufficiency in a 2-year-old-boy, treated by H.C. Stansel in New Haven, where the aortic valve was closed and the valved conduit carried the entire cardiac output. Also mentioned are the canine experiments of Sarnoff and associates who totally and permanently occluded the ascending aorta and thus diverted the entire cardiac output through a valved conduit to the descending thoracic aorta without apparent impairment of the circulation. That is, the animals were observed postoperatively to run, jump, and swim. However, in cases of supravalvular stenosis, Cooley and Norman[3] indicate that reduced aortic flow may adversely affect the coronary circulation.

CONCLUSIONS AND SUGGESTIONS

A flow model describing the fluid mechanics of left ventricular-aortic valved conduits has been established and shown to be useful in predicting conduit and aortic volumetric flow rates. The model can be used to investigate the effect of various design alterations on the performance of the valved conduit. For example, prosthetic valve selection can have a significant effect on the volumetric rate of flow through the conduit. The model may also be useful in analyzing other flow situations where a prosthetic valved conduit is employed in order to provide an alternate flow path. For example, the flow model, or portions of it, may prove useful in analyzing the fluid mechanics of left ventricular assist devices where blood is withdrawn from the left ventricle and its pressure is increased by an assist pump before its injection into the aorta.

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APPENDIX

In order to compare the relative magnitudes of the resistance terms in equation (3), the following parameter values were adopted:

Viscosity of blood, μ	0.033 poise
Length of aorta, L_a	25 cm.
Diameter of aorta, D_a	27 mm.
Diameter of stenotic aortic valve, D_{av}	12.1 mm.
Length of conduit, L_c	20 cm.
Diameter of conduit, D_c	20 mm.
Aortic flow rate, Q_a	9.6 l/min.
Conduit flow rate, Q_c	6.4 l/min.
Aortic valve constant, C_{av}	$1{,}775 \frac{(\text{mm Hg})(\text{mm}^4)(\text{min}^2)}{1^2}$
Conduit valve constant, C_{cv}	$25{,}300 \frac{(\text{mm Hg})(\text{mm}^4)(\text{min}^2)}{1^2}$

Substituting these values into equations (4), (5), (10) and (11) gives

$R_a = 0.8 \times 10^{-5}$ PRU
$R_c = 2.1 \times 10^{-5}$ PRU
$R_{av} = 79.5 \times 10^{-5}$ PRU
$R_{cv} = 101.2 \times 10^{-5}$ PRU,

where PRU stands for peripheral resistance units, (mm Hg) (min) (ml^{-1}), according to Strandness and Sumner.[13] Based on these results, it can be concluded that R_a and R_c are relatively small, compared to the valvular resistances, and thus can be neglected in equation (3).

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